

## Strength training reduces arterial blood pressure but not sympathetic neural activity in young normotensive subjects

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**Carter, Jason R., Chester A. Ray, Emily M. Downs, and William H. Cooke.** Strength training reduces arterial blood pressure but not sympathetic neural activity in young normotensive subjects. *J Appl Physiol* 94: 2212–2216, 2003. First published January 31, 2003; 10.1152/jappphysiol.01109.2002.—The effects of resistance training on arterial blood pressure and muscle sympathetic nerve activity (MSNA) at rest have not been established. Although endurance training is commonly recommended to lower arterial blood pressure, it is not known whether similar adaptations occur with resistance training. Therefore, we tested the hypothesis that whole body resistance training reduces arterial blood pressure at rest, with concomitant reductions in MSNA. Twelve young [ $21 \pm 0.3$  (SE) yr] subjects underwent a program of whole body resistance training 3 days/wk for 8 wk. Resting arterial blood pressure ( $n = 12$ ; automated sphygmomanometer) and MSNA ( $n = 8$ ; peroneal nerve microneurography) were measured during a 5-min period of supine rest before and after exercise training. Thirteen additional young ( $21 \pm 0.8$  yr) subjects served as controls. Resistance training significantly increased one-repetition maximum values in all trained muscle groups ( $P < 0.001$ ), and it significantly decreased systolic ( $130 \pm 3$  to  $121 \pm 2$  mmHg;  $P = 0.01$ ), diastolic ( $69 \pm 3$  to  $61 \pm 2$  mmHg;  $P = 0.04$ ), and mean ( $89 \pm 2$  to  $81 \pm 2$  mmHg;  $P = 0.01$ ) arterial blood pressures at rest. Resistance training did not affect MSNA or heart rate. Arterial blood pressures and MSNA were unchanged, but heart rate increased after 8 wk of relative inactivity for subjects in the control group ( $61 \pm 2$  to  $67 \pm 3$  beats/min;  $P = 0.01$ ). These results indicate that whole body resistance exercise training might decrease the risk for development of cardiovascular disease by lowering arterial blood pressure but that reductions of pressure are not coupled to resistance exercise-induced decreases of sympathetic tone.

weight training; hypertension; neural control; muscle sympathetic nerve activity; resistance training

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HYPERTENSION IS A PRIMARY risk factor for the development of coronary heart disease and stroke, which are the first and third leading causes of death in the United States. Health organizations such as the American Heart Association and the American College of Sports Medicine have endorsed aerobic exercise as a means for preventing or controlling hypertension, but

only recently has resistance training been similarly endorsed (1). There is general agreement in the literature that aerobic training elicits small reductions of arterial blood pressure, but evidence from resistance training studies is not as clear. Data from cross-sectional studies reveal that weight lifters either have elevated (20), reduced (21), or similar (5, 13) arterial blood pressures compared with untrained controls. Data from longitudinal studies suggest that resistance training either decreases (8, 9, 18, 22, 24) or does not change (9, 19, 23) arterial blood pressure at rest.

The mechanisms responsible for changes of arterial blood pressure after physical training appear to be numerous and have not been clearly defined, but aerobic exercise training has been shown to decrease resting plasma norepinephrine levels (3) as well as renal sympathetic nerve activity (12) and muscle (7) sympathetic nerve activity (MSNA). Although some studies have reported reductions of arterial blood pressure after resistance training (8, 9, 18, 22, 24), only one (18) has attempted to determine an underlying mechanism. Ray and Carrasco (18) tested the hypothesis that resistance training reduces both arterial blood pressure and sympathetic outflow. However, they found that training-induced reductions of arterial blood pressure occur without significant reductions of MSNA. Of note, subjects performed isometric handgrip exercise training (18), which only engages an isolated portion of the muscular system. Whole body strength conditioning programs are considered to be an important component of a holistic fitness regimen and are being recommended for some patients with heart disease (15). Although resistance training programs may be beneficial in the treatment and control of hypertension (1), associations among whole body resistance training, arterial blood pressure, and arterial blood pressure control mechanisms have not been explored in detail.

On the basis of evidence from aerobic conditioning programs and the lack of mechanistic evidence from strength conditioning programs, it remains possible that whole body resistance training affects arterial blood pressure through adaptation of the sympathetic nervous system. Therefore, the primary goal of this

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study was to test the hypothesis that whole body resistance training reduces arterial blood pressure at rest and that these reductions are associated with reductions of MSNA.

## METHODS

**Subjects.** Twenty-two healthy men and three healthy women were recruited for this study. Individuals were screened and excluded from the training group if they had participated in any resistance training 1 mo before the study. All subjects were normotensive, nonsmokers, had no history of autonomic dysfunction, and passed a physical examination before entry into the study. For logistical reasons, subjects were enrolled into this study on a staggered basis, with some beginning either training or control periods 2–3 wk apart over ~18 mo. For this reason our design was not strictly randomized, but subjects were matched for age and recreational activity (questionnaire). Resistance training subjects ( $n = 12$ ; 11 men and 1 woman) were instructed to refrain from any other regular exercise workouts during the 8-wk study. Control subjects ( $n = 13$ ; 11 men and 2 women) were instructed not to alter their normal activity levels or include any heavy resistance training during the 8-wk study period. The protocol was approved by The Human Research Committee of Michigan Technological University, and all subjects gave written informed consent before participating.

**Measurements.** Subjects arrived at the laboratory after abstaining from caffeine and exercise for at least 12 h. The laboratory was maintained at temperatures between 22 and 24°C, and care was taken to make measurements at the same time of day for each subject. We ensured proper arm cuff size by aligning target marks indicating appropriate cuff size and recorded arterial blood pressure from the right arm with the wrist maintained at heart level and the subject supine. After 10 min of supine rest, arterial blood pressure was measured three consecutive times at 2-min intervals with an automated sphygmomanometer (arterial tonometry, Colin Medical Instruments, San Antonio, TX). Previous experience has revealed that the first blood pressure measurements taken with our automated sphygmomanometer are consistently high. The second and third measurements are stable. For this reason, we recorded the results of the third measurement rather than the average of all three. This procedure was used consistently for all subjects. The electrocardiogram and MSNA (Nerve Traffic Analyzer, model 662C-1, Dept. of Bioengineering, University of Iowa, Iowa City, IA) were recorded continuously during 5 min of supine rest. Data were sampled at 500 Hz and stored on computer with commercial hardware and software (WINDAQ, Dataq Instruments, Akron, OH).

MSNA was directly measured by inserting a tungsten microelectrode (Frederick Haer, Bowdoinham, ME) into a peroneal nerve at the popliteal fossa. A reference electrode was inserted subcutaneously 2–3 cm from the recording electrode. Both electrodes were connected to a differential preamplifier and then to an amplifier (total gain of 70,000), where the nerve signal was band-pass filtered (700–2,000 Hz) and integrated (time constant 0.1 s) to obtain mean voltage neurograms. Satisfactory recordings of MSNA were defined by spontaneous, pulse-synchronous bursts that increased during Valsalva staining and that did not change during tactile or auditory stimulation.

**Exercise testing.** Subjects reported to the training facility (Portage Wellness Center, Houghton, MI) at least 1 h postprandial. Maximal whole body muscular strength was tested before and after either 8 wk of heavy resistance strength

training ( $n = 12$ ) or 8 wk of active control ( $n = 8$ ) using the following seven isotonic exercises (Cybex Strength Systems, Medway, MA and Ground Zero, Colorado Springs, CO) (with the primary muscle groups trained shown in parentheses): leg press (quadriceps), leg curls (hamstring), chest press (pectoralis major and minor, deltoid, triceps), lateral row (trapezius, latissimus dorsi, rear deltoid), shoulder press (deltoid), bicep curls (biceps), and triceps extensions (triceps). The first set consisted of 10 warm-up repetitions. The second and third sets were performed with increased resistance to obtain approximately six to eight and four to six repetitions, respectively. One-repetition maximum (1 RM) values were calculated from the third set according to the following equation:  $1 \text{ RM} = \text{weight lifted}/[0.95 - (\text{number of repetitions} - 2)(0.025)]$  (6). Recovery time between sets and exercises was strictly controlled at 2-min intervals, and proper exercise form was taught and enforced by personal trainers. Arterial blood pressure and MSNA at rest, but not maximal whole body muscular strength, were measured prospectively before and after 8 wk of active control in 5 of the 13 control subjects.

**Exercise training.** Twelve subjects underwent three supervised resistance training sessions per week for 8 wk. During each training session, subjects completed three sets of seven isotonic exercises in the same order: leg press, leg curls, chest press, lateral row, shoulder press, biceps curls, and triceps extensions. The initial leg press machine (Cybex Strength Systems) was unable to accommodate many of our subjects. The machine provided adequate resistance during the maximal strength test, but throughout the 8 wk of training subjects reached the machine's maximum resistance. A second leg press machine (Ground Zero) was also unable to accommodate many of our subjects. The remaining machines (Cybex Strength Systems) were used by all subjects. Subjects completed 3 sets of each exercise, performing 10 repetitions during sets 1 and 2 and as many repetitions as possible before concentric failure during set 3. Resistance was increased for the next session when subjects were able to complete at least 10 repetitions during the third and final set for each individual exercise. Recovery time between sets and exercises was strictly controlled at 2-min intervals. Training sessions lasted ~45 min. Trained assistants verbally encouraged the subjects and ensured proper form and technique.

**Data analysis.** Data were imported into a customized software program for analysis (WinCPRS, Absolute Aliens, Turku, Finland). R waves were detected and marked in the time series. Muscle sympathetic bursts were automatically detected from several criteria. Potential bursts were evaluated based on amplitude, using a signal-to-noise ratio of ~3:1, within a 0.5-s search window centered on a 1.3-s expected burst peak latency from the previous R wave (4). Mean burst peak latencies were calculated from the first iteration of the computerized burst detection, and a revised expected burst latency was entered for a second iteration. The procedure was repeated until further latency corrections failed to detect additional bursts or to change the average burst latency. Potential bursts were then displayed for visual conformation and edited by one of the investigators. MSNA was expressed as bursts per minute and bursts per 100 heartbeats.

**Statistical analysis.** All data were analyzed with commercial software (SAS Institute, Cary, NC). A two-way [group (resistance training vs. control)  $\times$  time (pretraining vs. post-training)] analysis of variance with repeated measurements on time was used to compare dependent variables of interest. It was determined a priori that significant group  $\times$  time interactions would be probed further with protected *t*-tests. Means were considered to be significantly different when  $P \leq$

Table 1. Maximum resistance for trained muscle groups before and after the exercise training and control periods

Exercise	Resistance Training		Control	
	Pretraining	Posttraining	Pretraining	Posttraining
Leg curl, kg	70 ± 3	82 ± 3*	62 ± 5	61 ± 5
Chest press, kg	73 ± 3	93 ± 3*	74 ± 10	72 ± 10
Lateral row, kg	74 ± 4	90 ± 4*	76 ± 4	77 ± 3
Shoulder press, kg	46 ± 2	62 ± 4*	47 ± 7	48 ± 8
Biceps curl, kg	39 ± 2	56 ± 3*	42 ± 6	43 ± 6
Triceps press, kg	65 ± 3	84 ± 5*	72 ± 8	72 ± 7

Values are means ± SE for 12 subjects in the resistance training group and 8 subjects in the control group. \*Significantly different from corresponding pretraining values,  $P < 0.01$ .

0.05. Results are expressed as means ± SE unless otherwise specified.

## RESULTS

**Subject characteristics.** Age, body weight, and height were  $21 \pm 0.3$  yr,  $82 \pm 4$  kg, and  $180 \pm 2$  cm for the resistance-trained subjects and were  $21 \pm 1$  yr,  $74 \pm 4$  kg, and  $178 \pm 4$  cm for the control subjects. Subject characteristics were not different between groups before and after the 8-wk training and control periods. Eight adequate nerve recordings from the 12 subjects assigned to the resistance training group both before and after the exercise training program, and 10 pretraining and posttraining nerve recordings from the 13 control subjects were successfully obtained.

**Resistance training.** Six of 12 subjects completed 100% of all training sessions as scheduled (3 days/wk for 8 wk). The remaining six subjects completed their 24 training sessions within 9 wk because of a holiday interruption. Subjects were instructed to complete a minimum of two workouts during the holiday on their own using the same training format with as closely matched machines as possible, but these sessions were not monitored by a trainer. For these six subjects, the training period was extended by 1 wk to ensure that each subject underwent 24 supervised training sessions.

Table 1 shows that resistance training increased strength in all muscle groups ( $P < 0.001$ ). Statistical comparisons pre- and posttraining for leg press were not applicable as a result of equipment problems as discussed previously. Percent increases for each of the remaining exercises were as follows: leg curl, 16.7%; chest press, 26.9%; lateral row, 22.1%; shoulder press, 34.7%; biceps curl, 42.5%; and triceps extension, 28.5%. Maximal strength did not change for subjects in the control group (Table 1).

**Arterial blood pressure and MSNA.** Blood pressures were not significantly different between resistance-trained and control subjects either before or after the 8-wk study period, but a significant interaction revealed decreases for subjects after resistance training ( $P = 0.009$ ). Figure 1 demonstrates that 8 wk of whole body resistance training significantly decreased resting systolic ( $130 \pm 3$  to  $121 \pm 2$  mmHg;  $P = 0.01$ ),

diastolic ( $69 \pm 3$  to  $61 \pm 2$  mmHg;  $P = 0.04$ ), and mean ( $89 \pm 2$  to  $81 \pm 2$  mmHg;  $P = 0.01$ ) blood pressures. Systolic ( $119 \pm 3$  to  $120 \pm 3$  mmHg;  $P = 0.6$ ), diastolic ( $64 \pm 2$  to  $62 \pm 2$  mmHg;  $P = 0.3$ ), and mean ( $82 \pm 2$  to  $81 \pm 2$  mmHg;  $P = 0.3$ ) blood pressures did not change over the course of the 8-wk study for subjects in the control group. Heart rate ( $65 \pm 3$  to  $63 \pm 2$ ) and MSNA were unaffected by 8 wk of resistance training (nerve data are presented in Table 2). Eight weeks of relative inactivity for subjects in the control group did not affect MSNA, but it significantly increased resting heart rate ( $61 \pm 2$  to  $67 \pm 3$  beats/min;  $P = 0.01$ ). Individual changes in systolic and diastolic blood pressures after resistance training are shown in Fig. 2.

## DISCUSSION

We evaluated arterial blood pressure and MSNA at rest in healthy, normotensive subjects before and after 8 wk of whole body resistance training. Our primary new finding is that resistance training effectively lowers arterial blood pressure without parallel reductions of MSNA. The data suggest that whole body resistive exercise decreases at least one primary risk factor for the development of coronary heart disease but eliminate reductions of MSNA as a possible underlying mechanism.

**Arterial blood pressure.** It has only been within the last several years that resistance training has been deemed safe for patients with cardiovascular disease (15) and considered potentially beneficial in controlling or reducing arterial blood pressure in patients with hypertension (1). Recommendations for lowering arterial blood pressure with strength training include using moderate weights, high repetitions, and brief rest periods to maximally stimulate the cardiovascular system (1). However, maximal strength gains are obtained by lifting heavy weights with few repetitions, separated by relatively long rest periods between sets (6). It is difficult to separate effects that may be due to either

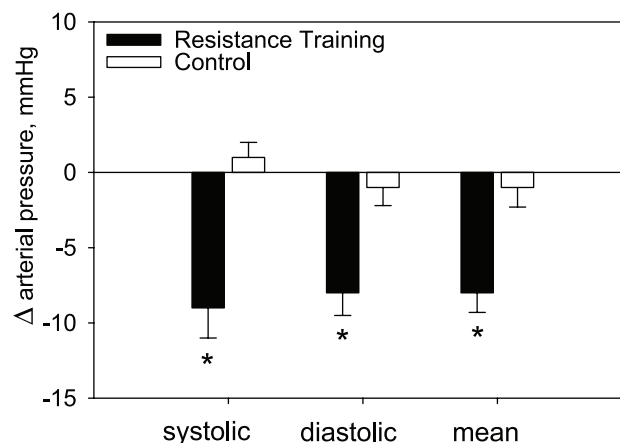


Fig. 1. Changes ( $\Delta$ ) in supine arterial blood pressures at rest before and after 8 wk of whole body resistance training or active control. Values are means ± SE for 12 subjects in the resistance training group and 13 subjects in the control group. \*Significantly different from corresponding pretraining values,  $P < 0.05$ .



Table 2. Muscle sympathetic nerve activity during 5 min of supine rest before and after exercise training

	Resistance Training		Control	
	Pretraining	Posttraining	Pretraining	Posttraining
MSNA				
Bursts/min	8 ± 1	9 ± 2	10 ± 1	10 ± 1
Bursts/100 heartbeats	11 ± 1	15 ± 3	17 ± 2	17 ± 3

Values are means ± SE for 8 subjects in the resistance training group and 10 subjects in the control group. MSNA, muscle sympathetic nerve activity;

muscular or cardiovascular adaptations, but when different resistance training studies are combined and analyzed with meta-analysis, the results strongly suggest that training with weights reduces both systolic and diastolic blood pressures (10, 11). In the present study, we sought to determine whether a resistance training program designed specifically to increase muscular strength without a significant cardiovascular component would effectively lower arterial blood pressure at rest.

Our data demonstrate that 8 wk of whole body high-intensity resistance exercise training significantly reduces systolic arterial blood pressure by 9 mmHg and diastolic and mean arterial blood pressures by 8 mmHg at rest. Collins et al. (2) reported that reductions of arterial blood pressure as little as 5 mmHg reduces the risk of stroke and myocardial infarction by 40 and 15% in hypertensive subjects. The small but significant reductions of arterial blood pressure at rest documented in the present study may not be of comparable clinical significance to reductions seen in hypertensive subjects, but we are aware of no studies that suggest such reductions are prognostically meaningless. Our data suggest that the addition of an 8-wk high-intensity strength training program to an active lifestyle not only substantially increases whole body muscular strength (Table 1) but also may decrease the risk for future development of cardiovascular disease in healthy young subjects.

**MSNA.** Although arterial blood pressure is not always affected by resistance training (19, 23), reductions have been documented after isometric handgrip (18, 24), circuit (8), and high-intensity (9, 22) strength training. Studies investigating the effects of resistance training on arterial blood pressure have been largely descriptive and have not attempted to identify underlying mechanisms. Evidence from aerobic conditioning programs suggests that exercise may decrease systemic sympathetic activity and arterial blood pressure in conjunction (3, 17), but until the present study, the effects of whole body high-intensity strength training on arterial blood pressure and MSNA had not been studied.

Human hypertension is associated with elevated baseline MSNA and the consequent effects of elevated norepinephrine on vascular smooth muscle tone (14). However, it does not necessarily follow that reductions

of arterial blood pressure after some intervention (including exercise) will occur with concomitant reductions of sympathetic nerve traffic. In the present study, reductions of MSNA did not occur after strength conditioning. MSNA expressed on a per-minute basis for 5 min was similar after, compared with before exercise training (Table 2). To account for potential differences in baseline heart rates between subjects (for example, in control subjects with elevated heart rates after the 8-wk study period), we also expressed MSNA as the number of bursts occurring during 100 cardiac cycles and detected no training effects. Our findings are in agreement with those of Ray and Carrasco (18), who documented reductions of arterial blood pressure after isometric handgrip training with no change in MSNA. Isometric handgrip training may be an effective non-pharmacological intervention for preventing and treating hypertension (18, 24), but participants do not benefit from whole body increases in muscular strength. Our results show that a more traditional strength conditioning program achieves both goals, increased muscular strength and decreased arterial blood pressure, but reinforce the concept that the mechanisms underlying reductions of arterial blood pressure at rest do not depend on changes of MSNA. It should be noted that, although the microneurography technique is a

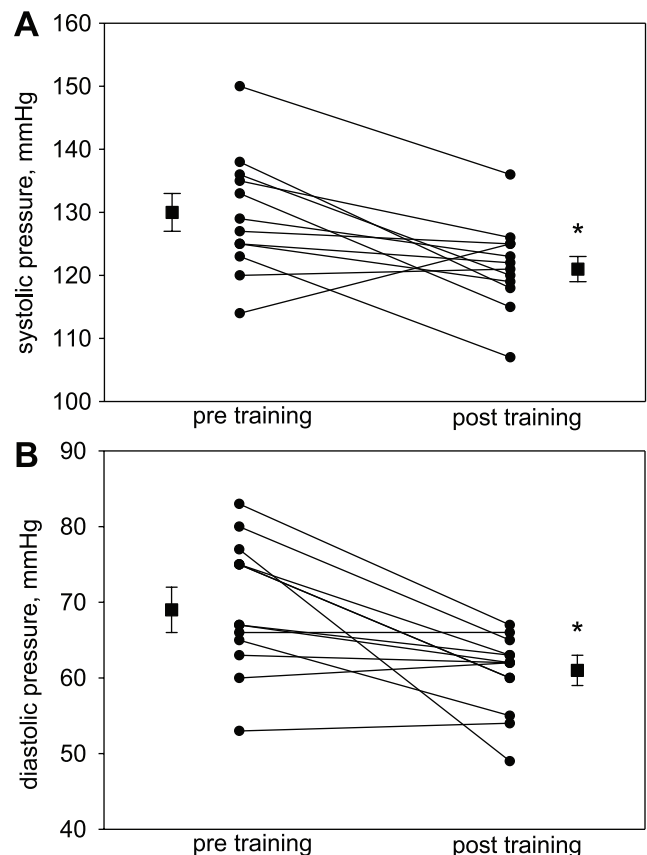


Fig. 2. Individual and mean (■ with error bars) changes in supine arterial blood pressures at rest before and after 8 wk of whole body resistance training. A: systolic pressure. B: diastolic pressure. \*Significantly different from corresponding pretraining values,  $P < 0.05$ .

powerful tool to investigate peripheral sympathetic activities to muscle or skin, it does not allow for extrapolation to other vascular beds, including the kidney. The kidney is important for long-term arterial blood pressure regulation (14), and endurance exercise training has been shown to decrease renal sympathetic nerve activity in rats (12) and humans (16).

**Limitations.** The results of the present study should be evaluated in light of at least two limitations. First, it remains possible that whole body strength training reduces both arterial blood pressure and MSNA but that reductions of MSNA were not detectable in our subjects. Average MSNA before training was only 8–10 bursts/min, and such low baseline activity may prohibit further reductions in response to exercise stimuli. Second, subjects enrolled in the present study were young, healthy, and normotensive. Longitudinal data support the concept that small reductions of arterial blood pressure protect against cardiovascular disease in hypertensive subjects, but corresponding long-term studies of asymptomatic normotensive subjects do not exist. We also made measurements with subjects in the supine vs. seated position, which might limit comparison of our results to other studies focusing on the clinical relevance of blood pressure reductions.

**Summary.** In conclusion, whole body resistance training effectively increases muscular strength and lowers arterial blood pressure at rest, but reductions of arterial blood pressure are not associated with reductions of MSNA. The data support the concept that resistance exercise training may be used as an exercise intervention to either control or lower arterial blood pressure and therefore may reduce at least one primary risk factor for the development of coronary heart disease. The data do not support the hypothesis that resistance exercise training directly affects baseline MSNA and therefore eliminate one major possible mechanism responsible for the antihypertensive effects of weight training.

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